



Bruxism – a review

Commander Ngoc-Nhung Tran, DC, USN and Captain John F. Johnson, DC, USN

Introduction

Many patients report to the dentist with pain in the preauricular region, difficulty in jaw opening, temporomandibular joint noise, and/or temporal headache upon awaking. They may also have been told by a family member that they clench or grind their teeth at night. When such patients consult a healthcare provider, they are often advised to wear an intraoral occlusal appliance to eliminate nocturnal parafunctional activity and reduce their pain. The purpose of this clinical update is to review the current understanding on the etiologic factors and mechanisms of bruxism and discuss management strategies.

Definition and epidemiology

Bruxism has been defined as an involuntary activity of the jaw musculature characterized by jaw clenching, bracing, gnashing and grinding of the teeth while asleep.¹ According to the International Classification of Sleep Disorders, sleep bruxism can be classified as a parasomnia, a group of sleep disturbances that also includes sleep walking, nightmares, sleep talking and enuresis.² The prevalence of bruxism ranges from 14-20% in children, 5-8% in adults but decreases to 3% in those 60 and over.^{3,4} There is no gender predilection.⁵ One in 5 patients with bruxism reports concomitant orofacial pain complaints.⁶

Etiologic factors and mechanisms:

Historically, occlusal discrepancies and dental-skeletal anomalies were hypothesized as major causes in the initiation and perpetuation of bruxism. However, current understanding does not support these conditions as etiologic factors.^{7,8} The association between bruxism and sleep came from studies using polysomnography in which masticatory motor activity, autonomic parameters and brain electrical activity (EEG) were recorded during sleep.⁹ Such investigations indicated that bruxism, as measured by increased electromyographic (EMG) activity in the masticatory muscles, is the result of an arousal response or a sudden change in the depth of sleep during which an individual transitions from deeper to lighter sleep.⁸ Although bruxism can occur in all stages of sleep, it is more often observed during stage 1 and 2 of non-rapid eye movement (NREM) sleep and during rapid eye movement (REM) sleep.⁹ Other physiologic

changes observed during the sleep arousal response include gross body movements, i.e. involuntary leg movement or turning, appearance of K complexes (single, biphasic large amplitude spikes) in the EEG, increases in heart rate, altered respiratory patterns, and peripheral vasoconstriction.¹⁰ Although the exact neural mechanisms underlying repetitive masticatory muscle activity during sleep have not been elucidated, they may involve alterations in brain neurochemistry and inappropriate firing of the reticular activating system.⁹

Management strategies

The initial management of bruxism should be directed at identifying the causes of dysfunctional sleep and working toward reducing factors that might fragment the patient's sleep architecture. The following factors contribute to sleep disturbance:¹⁰

- Over the counter stimulants
- Excessive caffeine and alcohol consumption
- Cigarette smoking
- Stress
- Shift work, deployment schedule
- Pain, medical conditions
- Psychiatric disorders
- Others

Optimum sleep management will require active participation by the patient, and in some instances, may warrant an interdisciplinary approach with other health care providers. Non-pharmacologic treatments, such as cognitive behavioral therapy, can significantly reduce the physiologic arousal often present in patients with sleep complaints.¹¹ Sleep hygiene instructions should also be provided to patients.⁸

Although occlusal splints may be beneficial in protecting the dentition, the efficacy of intra-oral appliances in reducing nocturnal masticatory muscle activity and the report of pain upon awakening is unclear. Recently van der Zaag et al used polysomnography to examine the effects of splints on patients with sleep bruxism and found that approximately 66% of the subjects treated with appliances demonstrated either no change or an increase in masticatory muscle EMG activity.¹² To date, the amount of reduction in masticatory muscle activity required to produce a meaningful improvement in reported pain has yet to be quantified. Solely using an appliance to reduce the muscle activity associated with bruxism may not predictably result in an improvement of

symptoms.¹³ Comprehensive reviews by Dao et al and Kato et al conclude that splints are useful adjuncts in the management of sleep bruxism, but are not definitive treatment.^{14,15}

The short term use of pharmacotherapy aimed at reducing sleep onset, increasing sleep continuity and total sleep time has been helpful in managing sleep disturbances, including bruxism.⁸ Commonly prescribed drugs for this purpose include: muscle relaxants, benzodiazepines, and low dose of tricyclic antidepressants. Newer drugs approved for insomnia are the non-benzodiazepines such as zolpidem (Ambien®), zaleplon (Sonata®) and eszopiclone (Lunesta®). Unlike the benzodiazepines, which tend to suppress stages 3 and 4 NREM and REM sleep, the new non-benzodiazepines hypnotics may enhance stage 3 and 4 sleep, the restorative phases of sleep.¹⁵ The latest hypnotic drug approved by the FDA is ramelteon, a melatonin receptor agonist, but there are currently no published reports discussing its use in patients with bruxism.¹⁶

Summary

Bruxism should not be considered as an isolated, anatomic dental problem. Instead, it may be more accurately categorized as a sleep-related disorder with dental and masticatory muscle implications. The prognosis for bruxism is variable depending on the severity and duration of the factors producing arousals during sleep. Intraoral appliances may protect dental structures from wear; however, a long lasting reduction in nocturnal masticatory muscle activity and awakening pain complaints may not be possible without identifying and addressing the etiology of the underlying sleep disturbance.

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Dr. Tran is a second year fellow in the Orofacial Pain Center, and Dr. Johnson is the Chairman of the Orofacial Pain Center at the Naval Postgraduate Dental School.

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